Conferences and Reviews

A 15-Year-Old Girl With Acute Renal Failure
Clinicopathologic Conference

Discussants

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This discussion was selected from the weekly Grand Rounds in the Department of Pediatrics, University of California, San Diego, School of Medicine.

Case Presentation

VIVIAN M. REZNIK, MD: The patient, a 15-year-old girl, was admitted to University Hospital, San Diego, California, because for two months she had had nausea, vomiting, and malaise. Although the results of a routine urinalysis a year before her presentation had been normal, a urinalysis done by her private physician before admission showed 2+ glucose, 2+ protein, and numerous leukocytes. There was no history of recent analgesic or antibiotic administration.

On physical examination she was thin, pale, pubertal, and appeared chronically ill. Her weight was 46 kg (101 lb), height 63 cm (5 ft 3 in), and blood pressure 130/80 mm of mercury. She had shotty left anterior cervical nodes. On examination of her abdomen, her liver could be palpated 2 cm below the right costal margin. The spleen tip was palpable on deep inspiration. Her extremities showed no edema.

Laboratory values obtained on admission were as follows: serum sodium 139, potassium 3.3, chloride 102, and bicarbonate 16 mEq per liter; calcium, 2.50 mmol per liter (10.0 mg per dl); phosphorus, 2.07 mmol per liter (6.4 mg per dl); uric acid, 506 μmol per liter (8.5 mg per dl); cholesterol, 4.40 mmol per liter (170 mg per dl); and glucose, 5.1 mmol per liter (92 mg per dl); liver function test values were normal. Blood urea nitrogen concentration was 41.1 mmol per liter (115 mg per dl), and serum creatinine level was 610 μmol per liter (6.9 mg per dl). A urinalysis showed a specific gravity of 1.016, pH 6.0, 2+ protein, 1+ glucose, 0 to 50 leukocytes per high-power field, 0 to 2 coarse granular casts, and 2 to 5 granular casts.

Urine chemistry levels were as follows: sodium, 59 mmol per day (59 mEq per 24 hours); and potassium, 35 mmol per day (35 mEq per 24 hours). A complete blood count elicited the following values: hemoglobin, 84 grams per liter (8.4 grams per dl), hematocrit, 0.24 (24%); mean corpuscular volume, 82 fl (82 μm^3); mean corpuscular hemoglobin, 29 pg; mean corpuscular hemoglobin concentration, 350 grams per liter (35 grams per dl); leukocyte count, 13.7 × 10^9 per liter (13,700 per mm^3) with a normal differential; platelet count, 650 × 10^9 per liter (650,000 per mm^3); and erythrocyte sedimentation rate (ESR, Westergren), 125 mm per hour. A creatinine clearance was 0.15 ml per second (9 ml per minute) per 1.73 m^2, and the 24-hour urine protein excretion was 0.66 grams (660 mg). Total hemolytic complement and C3 complement levels were normal; an antinuclear antibody test was negative. A purified protein-derivative skin test was negative. Chest x-ray and hand films were normal. An intravenous pyelogram (IVP) with tomography showed decreased uptake with normal kidney size (right kidney 12.7 cm, left kidney 13.0 cm; normal, ± 2 standard deviations = 11.8 ± 3.0 cm) and no signs of obstruction. Cultures of blood and urine were negative for bacterial or viral pathogens.

Differential Diagnosis

MONICA RIECKHOFF, MD: This is an interesting case of a 15-year-old female adolescent with renal failure that raised several perplexing issues. The first problem was to determine whether her renal failure was acute or chronic. The next question was whether prerenal, renal, or postrenal effects (or all three) were to blame. Finally, if her problem was intrarenal, were there any clues about what disease process was occurring?

First, we should consider the time course of her renal failure. Did she have acute renal failure, acute worsening of chronic renal failure, or chronic renal disease? Differentiating among these is important to target possible causes.

This patient was young and would be expected to tolerate a substantial reduction in renal function before presenting with uremic symptoms such as nausea, vomiting, and malaise. These symptoms tend to occur when the glomerular filtration rate is less than 5% to 10% of

(Rieckhoff MJ, Newbury RO, Kung FH, Reznik VM: A 15-year-old girl with acute renal failure—Clinicopathologic conference. West J Med 1995; 162:250-254)

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ABBREVIATIONS USED IN TEXT

ESR = erythrocyte sedimentation rate
IVP = intravenous pyelogram

normal. Her creatinine clearance was about 10% of normal, which would overestimate her glomerular filtration rate because there is tubular secretion of creatinine. Patients, particularly children with chronic renal failure, may tolerate a blood urea nitrogen concentration of greater than 35.7 mmol per liter (100 mg per dl) and a serum creatinine concentration of greater than 884 μmol per liter (10 mg per dl) without symptoms.

Another helpful piece of information was her normal urinalysis one year before she was seen. Although not conclusive, it suggests that she had no severe dysfunction of her kidneys at that time. The pronounced degree of renal failure less than a year later suggests a fairly rapid process.

The patient was well until two months before presenting and, although appearing chronically ill, had only a mild discrepancy between her height (40th percentile) and weight (20th percentile), suggesting mild weight loss without notable effect on stature.

Looking at her laboratory data, a normal potassium level could have several explanations: There was time for renal and extrarenal adaptation, anorexia had resulted in decreased potassium intake, or a renal defect exists in potassium handling. An elevated phosphorus level with normal calcium values and no evidence of renal osteodystrophy on roentgenography suggests insufficient time for compensatory hyperparathyroidism or altered vitamin D metabolism, which is seen in patients with chronic disease. Normocytic anemia could be found in acute renal disease from hemolysis, as seen in patients with the hemolytic-uremic syndrome, renal vein thrombosis, or systemic lupus erythematosus. It would have been helpful to know if there was evidence of hemolysis on the smear with burr cells, helmet cells, or schistocytes and to obtain a reticulocyte count. In addition, several systemic diseases associated with acute renal failure cause nonhemolytic normocytic anemia. In patients with chronic renal disease, anemia is caused by decreased erythropoietin production and the increased erythrocyte destruction that can result in hematocrits of 0.20 to 0.25 (20% to 25%), but this usually takes place over a period of months. Finally, the finding of normal rather than small kidney size on IVP suggests a more acute process. Normal-sized kidneys, however, can be seen in chronic diseases such as polycystic kidney disease, amyloidosis, and scleroderma. Although the patient presents a mixed picture, the findings suggest a relatively acute process and the possibility that her nausea, vomiting, and malaise were due to an underlying disease rather than uremia.

The next issue is whether there is evidence of prerenal or postrenal causes of renal failure. Prerenal disorders include problems with renal perfusion from cardiac failure, volume depletion, shock, or renovascular disease. We are missing some key pieces of information in the patient's physical examination, and I assume their absence reflects their normality. These include her pulse, temperature, or signs of heart failure (liver down 2 cm, but no jugular venous distension or edema). More details on her mental state and perfusion would have been helpful. She was not hypotensive. Patients with prerenal disorders typically present with a low fractional excretion of sodium whereas those with acute tubular necrosis or tubulointerstitial disease present with an increased fractional excretion of sodium, as seen in this patient. Prerenal factors, therefore, seem unlikely to be playing a major role. Postrenal causes are safely ruled out by the lack of obstruction on IVP.

How was the renal parenchyma involved? Her urinalysis showed glucosuria, suggesting tubular dysfunction. The proteinuria—0.66 grams per day (660 mg per 24 hours)—is notable, but is not in the nephrotic range. A normal cholesterol level and lack of edema also are against the nephrotic syndrome. A serum albumin level might have been helpful. The proteinuria could be due to tubular or glomerular dysfunction. Electrophoresis of the urine might show low-molecular-weight proteins, suggesting isolated tubular dysfunction. The leukocytes in the urine could be from a urinary tract infection, glomerular disease—either proliferative or inflammatory—or tubulointerstitial disease and can be seen in rare cases of urolithiasis or tumor. Staining for eosinophils would have been helpful to evaluate for acute interstitial nephritis, particularly from medications. The absence of hematuria makes glomerulonephritis less likely. Granular casts that consist of coarse or fine particles embedded in hyaline matrix are seen in normal healthy persons, but can be seen in persons with chronic lead intoxication, dehydration, glomerulonephritis, and tubulointerstitial disease. The patient has acidosis with an increased anion gap, which could be from lactate, ketoacids, salicylates, ethylene glycol or methanol ingestion, or simply uremia with a decreased ability to excrete organic acids. There are insufficient data to exclude renal tubular acidosis or Fanconi's syndrome, which over time can cause renal failure through secondary complications such as nephrocalcinosis.

An interesting point is that the patient's degree of renal insufficiency is more severe than commonly seen in patients with isolated tubular disease in whom tubular dysfunction is usually more prominent than the reduction in the glomerular filtration rate. There is no history of polyuria or nocturia to suggest tubular concentrating problems. The IVP showed decreased uptake, which could be from glomerular or tubulointerstitial disease, with pressure backing up because of luminal obstruction. Overall, the picture suggests substantial tubulointerstitial disease, but glomerular disease cannot be excluded.

Her history includes some other important information. She had two months of nausea, vomiting, and malaise, which may simply be from her azotemia or

could be from another underlying illness. In addition, she had borderline hypertension (90th to 95th percentile), shotty cervical adenopathy, possible hepatosplenomegaly, a markedly increased sedimentation rate, mildly elevated leukocyte count with a normal differential, pronounced normocytic anemia, and normal liver function test values.

The differential diagnosis of acute renal failure is extensive in this patient, given her nonspecific presenting features. Developmental abnormalities such as cystic disease, renal hypoplasia, or dysplasia are ruled out by a normal IVP. Tumors can cause renal disease through direct invasion of the kidney, but enlarged or distorted kidneys would be expected to be seen on IVP. Tumors such as lymphoma would certainly explain this patient's associated symptoms, hepatosplenomegaly, and laboratory findings. Also, an immune response to tumor antigen can cause disease, but most commonly it causes membranous glomerulopathy or, in the case of lymphoma, minimal change disease. Uric acid nephropathy occurs most commonly in patients being treated for malignant neoplasms, and this patient's uric acid level is only mildly elevated.

Metabolic diseases commonly affect the kidneys. Many of these diseases are part of syndromes that include developmental delay and other features not present in this patient.

Acute tubular necrosis certainly causes acute renal failure, but its presence would not explain the two-month history of symptoms and pronounced anemia. Heavy metal poisoning is consistent with her history, and it would be important to probe for any possible exposure, particularly to lead, arsenic, or mercury. A urine screening test for heavy metals should be done. Even more common would be medication-related acute tubular necrosis, but there was no history of medication use.

Intravascular coagulation can result from renal vein thrombosis; however, this is typically seen in infants with sepsis and shock or in children with the nephrotic syndrome or cyanotic heart disease. The kidneys are usually tender and enlarged with gross or microscopic hematuria. Cortical necrosis is seen in patients with the hemolytic-uremic syndrome, but they have thrombocytopenia. This patient has a thrombocytosis. The clinical course in this patient is also not consistent with this diagnosis.

Glomerulonephritis is unlikely given the history and lack of hematuria. Patients with poststreptococcal glomerulonephritis usually have a preceding upper respiratory tract or skin infection one to two weeks before their illness, a low C3 level, and hematuria with erythrocyte casts. The kidneys tend to be enlarged. This diagnosis could be ruled out with antistreptolysin O and streptozyme titers. Another possibility is membranoproliferative disease, but hematuria is usually present, and C3 and total complement levels are reduced in about 40% of patients. Patients with idiopathic rapidly progressive nephritis tend to have edema, gross hematuria, hypertension, anemia, and hypergammaglobulinemia. Acute renal failure often develops after an acute nephritic or nephrotic episode. Finally, membranous glomerulopathy is uncommon in children and usually presents as the nephrotic syndrome. Almost all patients have microscopic hematuria and progress to renal failure over many years. Glomerulonephritis can be due to endocarditis, but this patient had no murmur or history of fevers.

Connective tissue disease is a concern, given the systemic symptoms and increased ESR. Patients with systemic lupus erythromatosus typically have a high ESR and other symptoms such as fever, rash, arthritis, and lung, heart, or central nervous system disease, but occasionally kidney disease may be the only manifestation. It cannot be ruled out, but is unlikely in this patient, given the negative antinuclear antibody test. A lupus erythematosus preparation might also be useful. Juvenile rheumatoid arthritis in a young woman this age would probably present as polyarthritis of the knees, wrists, ankles, or elbows. In addition, there is usually hematuria and the kidneys are enlarged. Scleroderma is a rare disease in childhood, and renal disease occurs primarily in patients with the systemic form, which may antedate skin disease. Progressive renal disease rarely develops. Other autoimmune diseases are unlikely, given the lack of associated symptoms.

Acute tubulointerstitial nephropathy is a category of renal parenchymal disease that best fits the time course and laboratory findings in this patient.[1-3] There are many different causes of this disease.

The use of drugs, particularly nonsteroidal anti-inflammatory drugs, should be considered if there is any history of long-term use. Nonsteroidal anti-inflammatory drugs, unlike other medications causing tubulointerstitial nephropathy, only rarely cause hypersensitivity reactions and hematuria, but edema is usually prominent.

Pyelonephritis is unlikely to be present when cultures are negative. Other systemic infections, however, should be considered.[4] The first cases of tubulointerstitial nephritis associated with scarlet fever occurred in the 1860s. Since then, many other infections have been added to the list. Streptococcal infection is unlikely because the patient's symptoms would be more acute, with evidence of streptococcal infection, and the kidneys are often enlarged.[5] Other diseases that are also unlikely because of their more rapid onset (from days to a few weeks), associated symptoms (such as diarrhea, fever, pneumonia), and typically a travel history or exposure include those caused by *Staphylococcus* species, gram-negative bacilli, typhoid fever, diphtheria, brucellosis, legionellosis, leptospirosis, *Mycoplasma* species, *Campylobacter* species, and endocarditis.[6,7] Many of these could be ruled out by titer or culture.

Viral infections and tubulointerstitial nephropathy were first reported with measles, although these cases were severe, complicated by streptococcal infection, and resulted in death. Epstein-Barr virus infection is a consideration in this patient. Typically, however, patients have a more abrupt onset, with fever, pharyngitis, rash,

adenopathy, and hepatosplenomegaly.[8,9] Her age and other symptoms fit nicely, however. Liver function tests may show mildly abnormal values, and blood tests may show atypical lymphocytosis, hematuria, and hemolytic anemia with a positive Coombs' test and, rarely, thrombocytopenia and agranulocytosis. There are, however, reports of cases without fever, pharyngitis, or atypical lymphocytes and that present over the course of two months. Epstein-Barr virus infection is certainly a concern, and a Monospot test and Epstein-Barr virus titers are warranted. Cytomegalovirus is usually associated with transplantation or other immunocompromise or in infants. Symptoms would be similar to those of infectious mononucleosis with possible hemorrhagic cystitis. A urine test for cytomegalovirus could be done. Infection with the human immunodeficiency virus can involve the kidney with substantial proteinuria, but patients usually have a history of other human immunodeficiency virus-related illnesses.[10] Hantavirus, only recently described here in the United States, presents with fever and hemorrhage. Serum hepatitis has associated arthralgias, arthritis, urticaria, and rash.

Other infections associated with tubulointerstitial nephritis include Rocky Mountain spotted fever, schistosomiasis, and malaria, but these also cause more severe illness and have associated symptoms. Toxoplasmosis is a febrile illness with malaise, cervical adenopathy, hepatosplenomegaly, rash, and uveitis, and systemic disease is usually seen in infants and immunocompromised patients. There is, however, a case of a 10-year-old girl with uveitis and tubulointerstitial nephropathy who was otherwise well.[11] Chorioretinitis is commonly not recognized. Toxoplasmal titers should be checked, and it may be possible to see organisms on renal biopsy. Tuberculosis is possible, despite the negative purified protein-derivative test, if the patient is anergic. The IVP and normal chest roentgenogram, however, are against a diagnosis of tuberculosis. Secondary syphilis is a consideration, although renal involvement is uncommon and usually appears as membranous glomerulopathy.[12] Often patients have a rash (palms and soles), mucous membrane involvement, fever, and arthralgias. A rapid plasma reagin or VDRL test should be done, and it may be possible to find spirochetes on dark-field examination. Fungal disease should be considered but is unlikely in a patient without immunocompromise.

Besides infection, acute tubulointerstitial nephritis can be seen with glomerulonephritis and collagen vascular diseases, which have already been discussed. Necrotizing systemic vasculitis is possible, but polyarteritis nodosa often produces systemic vasculitis with abdominal pain, fever, and hematuria. In patients with small-vessel vasculitis, purpura, fever, and eosinophilia are commonly present.

In patients with hereditary tubulointerstitial nephritis, also called medullary cystic disease and familial juvenile nephronophthisis, the kidneys are small with severe diffuse cortical atrophy and cysts at the corticomedullary junction. Children usually fail to thrive and have polydipsia and polyuria.

Some of the systemic diseases associated with tubulointerstitial nephropathy are more chronic but also possible candidates in this case. Sarcoidosis is a disease of noncaseating granulomas involving the lungs, liver, spleen, bone marrow, and lymph nodes.[13,14] It is more prevalent in African Americans and Hispanics, men, and older patients. Most infected children have hilar adenopathy, hepatosplenomegaly, and cervical adenopathy. Hypercalcemia, modest proteinuria, sterile pyuria, and anemia are common in patients with renal involvement.

Dysproteinemia and neoplasms such as Waldenström's macroglobulinemia are rare in children and would cause associated symptoms, as would cryoglobulinemia. Occasionally tubulointerstitial nephropathy is seen as part of the renal involvement in neoplasms such as lymphoma and leukemia.[15-18] Usually there is an associated glomerulopathy causing a greater degree of proteinuria than was seen in this patient, but it should be included in the differential diagnosis.

Finally, idiopathic tubulointerstitial nephritis can be associated with anti-tubular basement membrane antibody or immune complexes and also a syndrome of the disorder with uveitis. This syndrome was first described in 1975 and occurs in young women with similar systemic complaints as in this patient, anemia, and an elevated ESR.[19-21] Abdominal pain, muscle pains, fever, and kidney enlargement with substantial hypertension are sometimes present. On biopsy, besides the usual findings of tubulointerstitial nephritis, noncaseating granulomas can rarely be seen. Granulomas may also be found in the bone marrow. The uveitis usually does not appear for weeks to months after the renal disease.

Given the patient's nonspecific presenting features, many of the diseases mentioned could account for her relatively rapid renal failure, two-month history of nausea, vomiting, and malaise, anemia, and elevated ESR. It would be helpful to know by ultrasonography if she has hepatosplenomegaly. Considering the patient's age and sex, the syndrome of tubulointerstitial nephropathy with uveitis makes the best fit, and I would be concerned that uveitis may develop. Previously mentioned diseases must be ruled out and a renal biopsy obtained.

Clinical Diagnosis

Acute interstitial nephritis.

Clinical Course and Discussion

The diagnosis was not clear to the clinicians caring for this child. A bone marrow aspiration showed only myeloid hyperplasia. A gallium scan demonstrated intense uptake in the kidneys and several areas of abnormal uptake in lymph nodes. Biopsy specimens of a left scalene node and a right femoral node both showed a nodular tumor with a mixture of lymphocytes and large atypical histiocytes that were compatible with Reed-Sternberg cells. An initial diagnosis of nodular sclerosing Hodgkin's lymphoma was made. The patient under-

went exploratory laparotomy for staging and a kidney biopsy. Tumor nodules were seen on sections of abdominal lymph nodes, liver, and spleen. There were no nodules on the surface of the kidneys. There was no ureteral obstruction. The histologic findings were considered consistent with a mixed-cellularity type of Hodgkin's lymphoma, stage IVB.[22]

Light microscopy of the kidney showed diffuse infiltration of lymphocytes and plasma cells throughout the interstitium. The glomeruli showed a mild increase in mesangial matrix. Immunofluorescence studies of the kidney showed irregular, segmental, granular deposition of complement C3 in the mesangial region of most glomeruli.[22]

A regimen of intravenous hydration and chemotherapy was begun. The patient received methotrexate, vincristine sulfate (Oncovin), and procarbazine hydrochloride with daily oral prednisone (MOPP). After the first two weeks, her blood urea nitrogen concentration was 8.6 mmol per liter (24 mg per dl), and the serum creatinine concentration was 160 μmol per liter (1.8 mg per dl). Monthly chemotherapy with the same agents was continued for six months. At that time, her urea nitrogen concentration was 6.8 mmol per liter (19 mg per dl), serum creatinine 70 μmol per liter (0.8 mg per dl), and the results of a urinalysis were normal. The IVP was repeated and was normal, showing reduced renal size. A kidney biopsy was again done and showed a pronounced decreased interstitial infiltrate. She was treated with monthly courses of chemotherapy for a total of 18 months. After this, she received vinblastine sulfate weekly for two years. She has been in remission for 12 years, and her renal function remains normal.

This was an unusual case of lymphoma presenting as acute renal failure. Malignant neoplasms can present as acute renal failure, acute glomerulonephritis, the nephrotic syndrome, or asymptomatic nephromegaly.[15-18,23-25] Pathologic disorders include minimal change nephrotic syndrome, membranoproliferative glomerulonephritis, and membranous glomerulonephritis. This case was unusual because there was direct infiltration of the interstitium, causing a mixed presentation of both glomerular and tubular abnormalities. The disease responded quickly and completely to chemotherapy, and the patient has done remarkably well over a long period of time.

REFERENCES

1. Koskimies O, Holmberg C: Interstitial nephritis of acute onset. Arch Dis Child 1985; 60:752-755

2. Ellis D, Fried WA, Yunis EF, Blau EB: Acute interstitial nephritis in children: A report of 13 cases and review of the literature. Pediatrics 1981; 67:862-870

3. Greising J, Trachtman H, Gauthier B, Valderrama E: Acute interstitial nephritis in adolescents and young adults. Child Nephrol Urol 1990; 10:189-195

4. Eknoyan G, Dillman RO: Renal complications of infectious diseases. Med Clin North Am 1978; 62:979-1003

5. Webster AD, Hall GH: Acute post-streptococcal toxaemic renal failure. Br Med J 1969; 4:665-666

6. Ooi BS, Chen BT, Tan KK, Khoo OT: Human renal leptospirosis. Am J Trop Med Hyg 1972; 21:336-341

7. Bain BJ, Ribush NT, Nicoll P, Whitsed HM, Morgan TO: Renal failure and transient paraproteinemia due to *Leptospira pomona*—Case reports and literature review. Arch Intern Med 1973; 131:740-745

8. Kopolovic J, Pinkus G, Rosen S: Interstitial nephritis in infectious mononucleosis. Am J Kidney Dis 1988; 12:76-77

9. Arm JP, Rainford DJ, Turk EP: Acute renal failure and infectious mononucleosis. J Infect 1984; 9:293-297

10. Strauss J, Abitbol C, Zilleruelo G, et al: Renal disease in children with the acquired immunodeficiency syndrome. N Engl J Med 1989; 321:625-630

11. Guignard JP, Torrado A: Interstitial nephritis and toxoplasmosis in a 10-year-old child. J Pediatr 1974; 85:381-382

12. Walker PD, Deeves EC, Sahba G, Wallin JD, O'Neill WJ: Rapidly progressive glomerulonephritis in a patient with syphilis—Identification of antitreponemal antibody and treponemal antigen in renal tissue. Am J Med 1984; 76:1106-1112

13. Bear RA, Handelsman S, Lang A: Clinical and pathological features of six cases of sarcoidosis presenting with renal failure. Can Med Assoc J 1979; 121:1367-1371

14. Cuppage FE, Emmott DF, Duncan KA: Renal failure secondary to sarcoidosis. Am J Kidney Dis 1988; 11:519-520

15. Moulin B, Ronco PM, Mougenot B, Francois A, Fillastre JP, Mignon F: Glomerulonephritis in chronic lymphocytic leukemia and related B-cell lymphomas. Kidney Int 1994; 42:127-135

16. Dabbs DJ, Striker LM, Mignon F, Striker G: Glomerular lesions in lymphomas and leukemias. Am J Med 1986; 80:63-70

17. Morel-Maroger Striker L, Striker GE: Glomerular lesions in malignancies. Contrib Nephrol 1985; 48:111-124

18. Rault R, Holley JL, Banner BF, el Shahawy M: Glomerulonephritis and non-Hodgkin's lymphoma: A report of two cases and review of the literature. Am J Kidney Dis 1992; 20:84-89

19. Dobrin RS, Vernier RL, Fish AL: Acute eosinophilic interstitial nephritis and renal failure with bone marrow-lymph node granulomas and anterior uveitis—A new syndrome. Am J Med 1975; 59:325-333

20. Vanhaesebrouck P, Carton D, De Bel C, Praet M, Proesmans W: Acute tubulointerstitial nephritis and uveitis syndrome (TINU syndrome). Nephron 1985; 40:418-422

21. Spital A, Panner JJ, Sterns RH: Acute idiopathic tubulointerstitial nephritis: Report of two cases and review of the literature. Am J Kidney Dis 1987; 9:71-78

22. McNeal RM, Mendoza SA, Kung FH, Griswold WR: Hodgkin's disease presenting as renal failure: Improvement in renal lesion and renal function with chemotherapy. Int J Pediatr Nephrol 1994; 2:99-101

23. Abouchacra S, Duguid WP, Somerville PJ: Renal cell carcinoma presenting as nephrotic syndrome complicated by acute renal failure. Clin Nephrol 1993; 39:340-342

24. Morris E, Phillips A, Wood BP: Radiological cases of the month: Presentation of lymphoma with renal insufficiency. Am J Dis Child 1993; 147:201-202

25. Hotta O, Taguma Y, Kurosawa K, Sudo K, Takahashi H: Membranous nephropathy associated with nodular sclerosing Hodgkin's disease. Nephron 1993; 63:347-350